

Atypical Atrioventricular Node Reciprocating Tachycardia Masquerading as Tachycardia Using a Left-Sided Accessory Pathway

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Objectives. The study was performed to document that atrioventricular node reciprocating tachycardia (AVNRT) can be associated with eccentric retrograde left-sided activation, masquerading as tachycardia using a left accessory pathway.

Background. The eccentric retrograde left-sided activation during tachycardia is thought to be diagnostic of the presence of a left free wall accessory pathway. However, it is not known whether AVNRT can occur with eccentric retrograde left-sided activation.

Methods. We studied 356 patients with AVNRT who underwent catheter ablation. Retrograde atrial activation during tachycardia and ventricular pacing were determined by intracardiac recordings, including the use of a decapolar coronary sinus catheter.

Results. The retrograde atrial activation was eccentric in 20 patients (6%). Eight of these patients had the earliest retrograde atrial activation recorded in the lateral coronary sinus leads, and 12 had the earliest retrograde atrial activation recorded in the

posterior coronary sinus leads, with the most proximal coronary sinus electrode pair straddling the coronary sinus orifice. These tachycardias were either the fast-slow or the slow-slow form of AVNRT. The slow-fast form of AVNRT was also inducible in 17 of the 20 patients. Successful ablation of the slow pathway in the right atrial septum near the coronary sinus ostium prevented the induction and clinical recurrence of reciprocating tachycardia in all patients.

Conclusions. Atypical AVNRT with eccentric retrograde left-sided activation was demonstrated in 6% of all patients with AVNRT masquerading as tachycardia using a left-sided accessory pathway. Ablation of the slow pathway at the posterior aspects of the right atrial septum resulted in a cure in these patients.

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It has been hypothesized that a dual atrioventricular (AV) conduction system is present in (1–3), and that functional longitudinal dissociation of the dual pathway can result in, reciprocating tachycardia (RT) (4–8). In typical atrioventricular node reciprocating tachycardia (AVNRT), the slow pathway (SP) conducts in the anterograde direction and the fast pathway (FP) in the retrograde direction. In atypical AVNRT, the FP conducts in the anterograde direction, whereas the SP is used in the retrograde direction (9). Because the FP is located near the interatrial septum and the SP is located near the coronary sinus (CS) (10), the earliest retrograde atrial activation is recorded in the interatrial septum in typical

AVNRT (11) and at the CS ostium in atypical AVNRT (10). In recent years, however, the traditional views of dual AV nodal pathways have been challenged. It is now generally believed that multiple pathways are present. Some of these pathways may involve perinodal regions that are outside the traditional SP and FP locations (12–17). Each pathway may be associated with a different form of AVNRT. However, the characteristics of these perinodal pathways have not been fully defined. Anatomic and histologic studies (18–20) have demonstrated that the perinodal region is heavily populated by the transitional cells. These cells are also present in the deeper layer of the atrial septum and may serve as the electric connection between the left atrium and the compact AV node. To determine whether these left-sided pathways are present, we inserted decapolar catheters in the CS to register retrograde atrial activation sequences during AVNRT. The results were used to test the following hypotheses: 1) There are multiple atrial inputs to the AV node. 2) Some of these pathways are located deep in the atrial septum or even in the AV groove along the left atrial free wall. 3) Because of the presence of these pathways, AVNRT may be associated with eccentric retrograde atrial activation sequences, masquerading as AV RT using a left free wall accessory pathway (AP). 4) Selective SP ablation can eliminate the AVNRT with eccentric retrograde atrial activation sequences.

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Abbreviations and Acronyms

AP	= accessory pathway
AV	= atrioventricular
AVNRT	= atrioventricular node reciprocating tachycardia
CS	= coronary sinus
FP	= fast pathway
RT	= reciprocating tachycardia
SP	= slow pathway
VA	= ventriculoatrial

Methods

Consecutive patients undergoing radiofrequency catheter ablation for all forms of AVNRT from January 1993 to February 1996 were included in the study. Written informed consent was obtained before the study. The electrophysiologic study and the radiofrequency catheter ablation were performed in the same session. All antiarrhythmic drugs had been discontinued for at least five half-lives. A 7F lumen decapolar catheter (Cordis-Webster) was inserted into the CS through the right internal jugular vein. The 10 poles on the decapolar catheter form five bipolar recording pairs. The distance between two bipoles was 2 mm, and the distance between two bipolar recording pairs was 5 mm. Bipolar pair 1 was the most distal pair and bipolar pair 5 was the most proximal pair. The proximal pair of electrodes was positioned at the CS ostium and the location was confirmed by a CS angiogram in all patients. In the earlier studies, only three or four bipolar pairs were connected to the recording system because of technical limitations. When three pairs were used, they were bipolar pairs 1, 3 and 5. When four pairs were used, they were bipolar pairs 1, 2, 4 and 5.

Figure 1 shows the catheter placement. Figure 1A shows the CS angiogram with the CS catheter in place. Once the CS catheter was positioned, the venous sheath was sutured or taped to the skin for fixation. The catheter was then taped to the sheath to maintain the same position throughout the study. Three 6F or 7F quadripolar catheters (Cordis-Webster) were introduced percutaneously through the femoral veins. The catheters were placed under fluoroscopic guidance to the high right atrium, AV junction and right ventricular apex for local

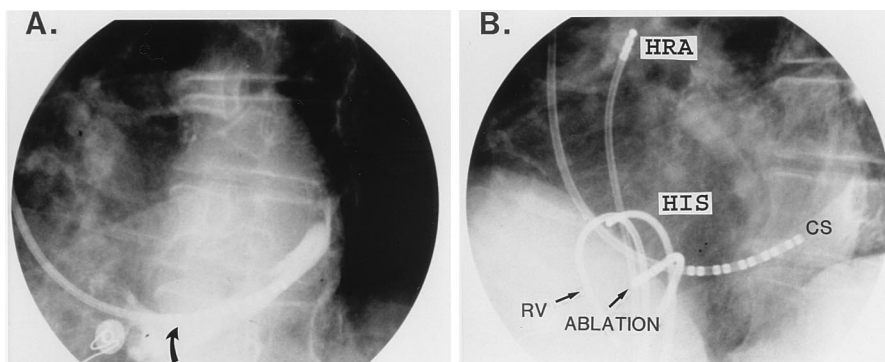
electrogram recording and pacing (Fig. 1B). The electrograms and fluoroscopic images were recorded by an Arrhythmia Research Technology CardioLab System. Cine films were also obtained in some patients to record the catheter locations. The electrogram signals were filtered at 30 to 500 Hz, and were digitized at 979 samples/s with 12 bits of accuracy. The activation times were determined by the usual criteria (21).

Standard pacing techniques were used at the high right atrium, CS and right ventricular apex to evaluate the antero-grade and retrograde dual AV node properties and the induction of RT. Isoproterenol (1 to 5 μ g/min) was used to facilitate the induction of AVNRT if baseline pacing failed to induce RT. In patients with an eccentric retrograde atrial activation sequence, adenosine (6 to 12 mg) was given during high right atrial and ventricular apex pacing and during RT. The results of this test were used to identify the site of block immediately before termination of RT, and also to create complete AV conduction block to demonstrate the absence of an adenosine-insensitive AP (22). Patients were excluded from the study if there was one or more features suggesting the presence of an AP (23), such as ventricular pre-excitation during atrial pacing, lengthening of the ventriculoatrial (VA) interval during bundle branch block, advancement of atrial activation by a premature ventricular contraction occurring during His bundle refractoriness, recording of an AP potential (24) or successful elimination of RT by delivering radiofrequency energy anywhere other than the low right atrial septum.

AVNRT was diagnosed if the following criteria were met: 1) There was no evidence for the presence of an AP by the criteria mentioned previously. 2) A narrow QRS complex RT was inducible by single or double premature atrial stimulation or rapid atrial pacing with or without isoproterenol infusion. 3) The RT could be terminated by rapid atrial or ventricular pacing. 4) SP ablation (25) prevented reinduction of RT with and without isoproterenol infusion. In most patients, there was an AV or VA “jump,” or both, defined by the prolongation of the AH or HA interval by 50 ms, whereas the S₁ to S₂ intervals were shortened by 10 ms, preceding the onset of RT (5,26,27). However, because a 50-ms jump does not have to occur in every patient with AVNRT (28), this was not used as a criterion for diagnosing AVNRT at our institutions.

The AVNRT was classified according to intracardiac re-

Figure 1. Catheter location. The image was recorded by cine film in the left anterior oblique projection. **A**, CS angiogram taken immediately after placement of the decapolar catheter. **Arrow** points to the location of the most proximal bipolar electrode, which was at the orifice of the CS. **B**, Location of all catheters at the time of successful SP ablation. **ABLATION** = ablation catheter; **HIS** = His Bundle electrogram; **HRA** = high right atrium; **RV** = right ventricle.



cordings into the slow-fast form (“typical”) (5,27), fast-slow form (“atypical”) (9,26,29) and slow-slow form (13), based on the AH and HA intervals measured at the His bundle electrode. The slow-fast form was diagnosed if the retrograde conduction during RT was associated with a short VA interval (<60 ms) (29,30) and the AH interval was longer than the HA interval. The fast-slow form was diagnosed if 1) the tachycardia was not initiated by a sudden (50 ms) jump of the AH interval (9,26); 2) the retrograde VA interval during RT was >60 ms (13); and 3) the HA interval was longer than the AH interval during the RT. The slow-slow form (13) was diagnosed if the anterograde conduction fulfilled the criterion of SP conduction, including a sudden jump of the AH interval at the initiation of RT, but the retrograde VA interval was >60 ms and the AH interval was greater than the HA interval during RT.

Determination of earliest retrograde atrial activation. The earliest retrograde atrial activation during RT and during ventricular pacing was determined by analyzing the times of atrial activation registered by intracardiac catheters. A concentric retrograde atrial activation sequence was thought to be present if the earliest retrograde A wave was registered either at the anterior atrial septum (His A) or at the posterior atrial septum (CS ostium A). An eccentric retrograde atrial activation sequence was thought to be present if the earliest atrial activation was registered inside the CS, at bipolar pairs 1 to 4 (electrodes 1 to 8) of the CS catheter.

Radiofrequency catheter ablation. A 7F quadripolar catheter (Cordis-Webster) with a 4-mm tip was used for mapping and for radiofrequency catheter ablation. Slow pathway ablation was performed using a standard right atrial septum approach, guided by the SP potential (25,31). In all patients, the mapping was performed in a stepwise fashion from the tricuspid annulus toward the CS ostium. We then gradually moved the catheter toward the mid-point between the His and CS ostium electrodes. When SP potentials were registered by the distal electrodes, 25 W energy was applied for 15 s. If an accelerated junctional rhythm was not observed, the energy application was stopped and mapping was continued. If an accelerated junctional rhythm was observed, the energy application was continued to complete a total of 60 s. Pacing was then performed to document the successful ablation of the SP and the inability to reinduce AVNRT. The end point of ablation was noninducibility of RT (25,31) by pacing at baseline and during isoproterenol infusion. The fluoroscopic image associated with a successful ablation site was recorded by computer or cine film. The patients were followed up monthly for the first 3 months either by telephone or outpatient visits, and thereafter only based on the recurrence of symptoms.

Statistical analysis. The unit of statistical analysis is the patient. Results are expressed as mean value \pm SD. Nonpaired *t* tests were used to compare the means of the electrophysiologic variables. A *p* value ≤ 0.05 was considered significant.

Table 1. Clinical Characteristics of 20 Study Patients

Male/female	4/16
Age (yr)	56 \pm 8
Range	34–72
Follow-up	14 \pm 7
RF energy applications	4 \pm 2
Recurrences	1

Data presented are mean value \pm SD, range or number of patients. RF = radiofrequency.

Results

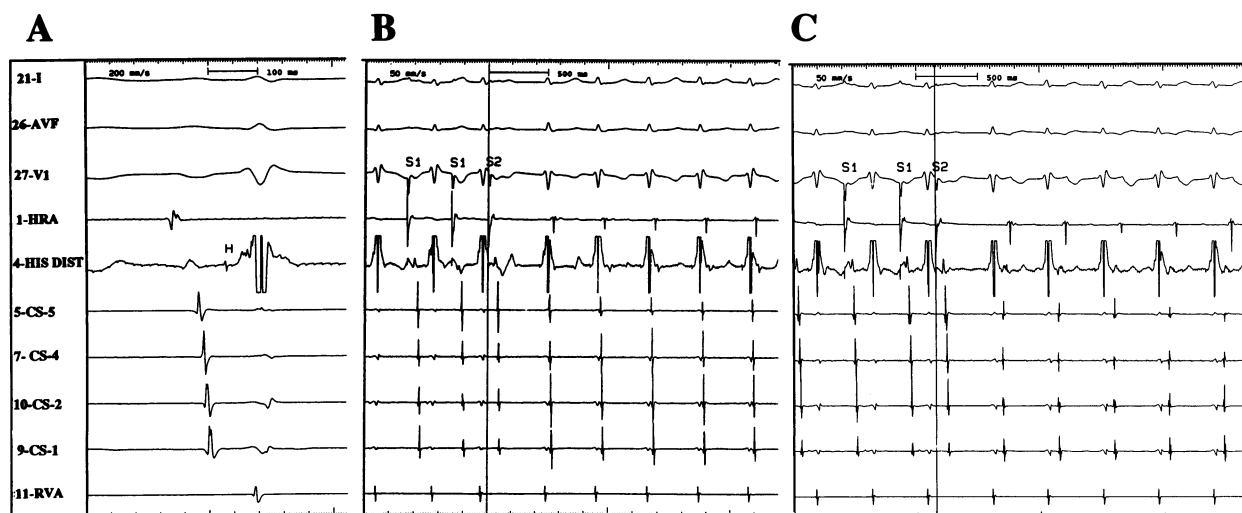
A total of 356 consecutive patients with AVNRT were included in the study. Of these, 310 patients had only the typical form of AVNRT, and 46 had at least one episode of the atypical form of AVNRT inducible during the study. Of the 356 patients, 20 had eccentric retrograde atrial activation sequences during either the slow-slow or fast-slow forms of AVNRT (Table 1). In 17 of these 20 patients, the classic (slow-fast) form of AVNRT with concentric retrograde atrial activation sequences was also inducible. There were significant differences of the electrophysiologic characteristics of these two kinds of AVNRT (Table 2). Transient left bundle branch block in 2 patients and right bundle branch block in 4 patients during RT did not change the VA interval or cycle length of RT. The P wave during the atypical form of AVNRT was usually negative in leads I, II and aVF and positive in lead V₁. There were no significant differences of P wave configurations between AVNRT with or without eccentric retrograde atrial activation sequences.

Only one patient had recurrence of RT 6 months after the procedure. Although both forms of AVNRT (slow-slow and slow-fast) were observed during the first electrophysiologic study, only the typical slow-fast form of AVNRT was inducible

Table 2. Electrophysiologic Characteristics of Typical Atrioventricular Node Reciprocating Tachycardia and Atrioventricular Node Reciprocating Tachycardia With Eccentric Retrograde Atrial Activation Sequences

	Typical AVNRT	AVNRT With Eccentric Retrograde Atrial Activation Sequences	<i>p</i> Value
Tachycardia CL (ms)			
Mean \pm SD	342 \pm 39	384 \pm 32	<0.005
Range	282–437	320–464	
AH interval (ms)			
Mean \pm SD	292 \pm 42	232 \pm 21	<0.001
Range	241–379	167–296	
HA interval (ms)			
Mean \pm SD	49 \pm 5	187 \pm 29	<0.001
Range	41–58	148–287	
RP interval (ms)			
Mean \pm SD	40 \pm 12	168 \pm 32	<0.001
Range	0–80	120–320	

AVNRT = atrioventricular node reciprocating tachycardia; CL = cycle length.



during the second study, with concentric retrograde activation sequences. The SP was again successfully ablated and the patient had no further recurrence of symptoms.

Eccentric retrograde atrial activation. Anterograde and retrograde dual AV node physiology was demonstrated in all 20 patients. In eight of the 20 patients, the earliest retrograde atrial activation in at least one episode of AVNRT was registered in bipolar pairs 1 and 2, corresponding to electrodes 1 to 4 of the decapolar CS catheter. These locations corresponded to the left atrial free wall. In the remaining 12 patients, the earliest retrograde atrial activation was registered in bipolar pairs 3 and 4, corresponding to electrodes 5 to 8 of the decapolar CS catheter, during at least one episode of AVNRT. These electrode locations were compatible with eccentric retrograde atrial activation in the posterior left atrium. The eccentric retrograde atrial activation sequences were associated with slow-slow AVNRT in 11 patients and with fast-slow AVNRT in nine patients.

Figures 2 and 3 show electrograms of our first patient with eccentric retrograde atrial activation sequences. Figure 2A shows that, during sinus rhythm, the atrial activation started from the high right atrium, then progressed to the His bundle region, followed by activations in the CS region. This finding is compatible with the correct electric connections of individual CS electrodes to the recording system. Figures 2B and 2C show the induction of slow-fast and slow-slow AVNRT, respectively, by atrial premature stimulation during the same study.

Figure 3 shows additional electrograms of the same patient shown in Figure 2. Figure 3, A and B, shows that during the same episode of RT, the earliest retrograde atrial activation changed from the middle electrodes (Fig. 3A) to the most distal two bipolar electrode pair (Fig. 3B) in the CS catheter. Therefore, the sites of earliest retrograde atrial activation were not fixed but moved from one place to another during the same study. The cycle lengths of the RT also changed but were not all due to the changes of the retrograde VA interval. The VA intervals during AVNRT with eccentric retrograde atrial activation

Figure 2. AVNRT. **A**, Normal activation sequences during sinus rhythm. **B and C**, Induction of the slow-fast and slow-slow forms of AVNRT, respectively, by atrial premature stimulation. The slow-slow form was associated with an eccentric retrograde atrial activation sequence (see also Fig. 3). HIS DIST = distal bipolar pair of the His bundle electrode; HRA = high right atrium; RVA = right ventricular apex. S1 = baseline stimulation; S2 = premature stimulation.

sequence in this patient as well as in the remaining 18 patients were all >60 ms. Figure 3C shows the retrograde atrial activation sequences during a different episode of RT. The activation was clearly eccentric, with the most distal CS electrodes recording the earliest activations. However, the bipolar electrogram configuration and the timing sequence among each other were different from that shown in Figure 3, A and B.

Figure 4A shows the activation sequences during catheter-induced ventricular tachycardia from the left ventricle in the same patient as shown in Figures 2 and 3. Figure 4B shows the retrograde activation sequences during left ventricular pacing after successful SP ablation in the same patient. In both panels there was retrograde VA Wenckebach conduction block. Left ventricular pacing was performed in the first three of the 20 patients and demonstrated VA conduction with Wenckebach cycle lengths of 425 to 500 ms.

When paced from the right ventricular apex, 14 patients had baseline VA conduction. The VA conduction was demonstrable in the remaining six patients during isoproterenol infusion. During S₁ to S₂ pacing from the right ventricular apex, 17 patients had decremental conduction due to HA interval prolongation, with eccentric retrograde atrial activation sequences. We did not observe HA dissociation during ventricular pacing. In these 17 patients, the retrograde atrial activation sequences were the same during pacing and during RT. In the remaining three patients, the retrograde atrial activation was less eccentric during ventricular pacing than during RT, and retrograde His was not seen during pacing. After ablation, the VA conduction remained eccentric but the

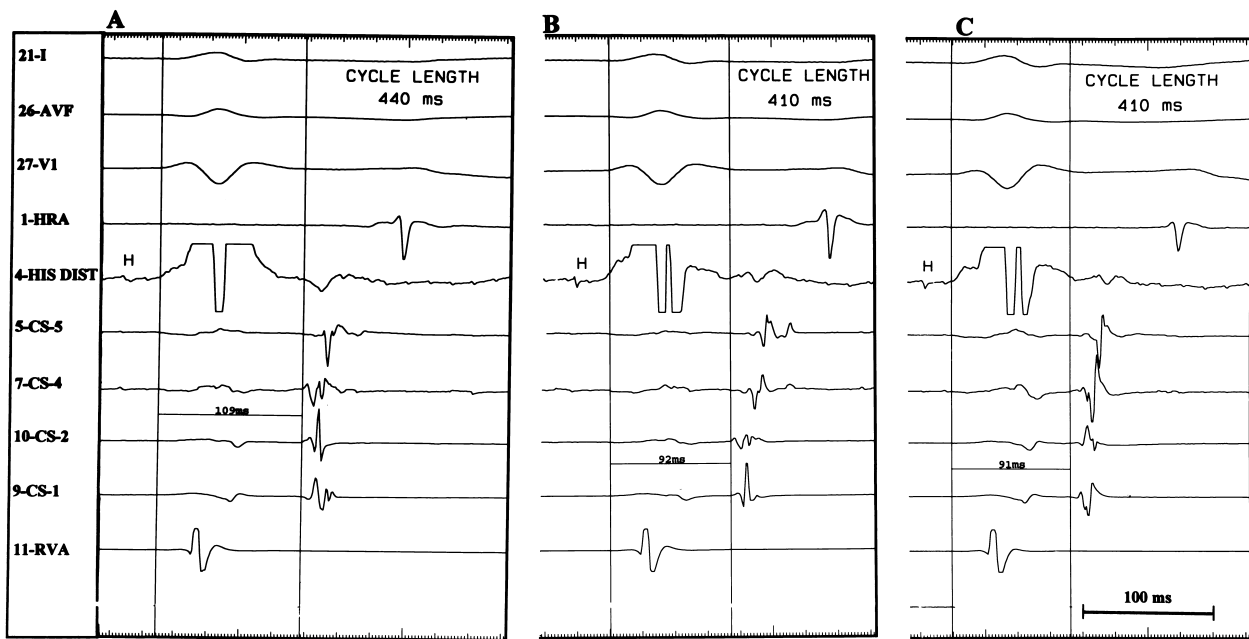


Figure 3. Eccentric retrograde atrial activation sequence during AVNRT in the same patient as that in Figure 2. **A**, During one episode of slow-slow AVNRT, the earliest retrograde atrial activation was in the mid-CS. **B**, The site of the earliest retrograde atrial activation moved to more distal CS sites during the same episode of tachycardia. **C**, During a different episode of tachycardia in the same study, the earliest retrograde atrial activation was at the distal CS. Abbreviations as in Figure 2.

VA Wenckebach cycle length was prolonged compared with baseline (from 368 ± 47 ms to 503 ± 58 ms, $p < 0.001$). Figure 5 shows the fluoroscopic image of the catheter location registered during the same study as that shown in Figures 2 to 4.

Figure 6 shows another example of eccentric retrograde atrial activation. Panel A shows that during sinus rhythm, the AH interval was 71 ms and the HV interval was 46 ms. Panel B shows the induction of sustained fast-slow AVNRT by single atrial premature stimulus. The earliest retrograde atrial activation was located in the distal CS (arrows). Slow pathway ablation prevented the recurrences of the RT in this patient. The CS angiogram during the study showed that the proximal CS electrodes were near the septum, whereas the distal electrodes were in the left ventricular free wall location. The retrograde atrial activation sequence during ventricular pacing remained eccentric after ablation.

In each patient, the location of the CS catheter during the study was confirmed by fluoroscopic images throughout the study. There was no significant dislocation of the catheter when the recordings were made. Premature ventricular contractions did not advance the atrial electrogram during RT, and adenosine resulted in complete VA and AV blocks. Except for the eccentric retrograde atrial activation sequence, there was no evidence to suggest the presence of a left-sided AP.

The patient shown in Figures 2 to 4 was the first patient

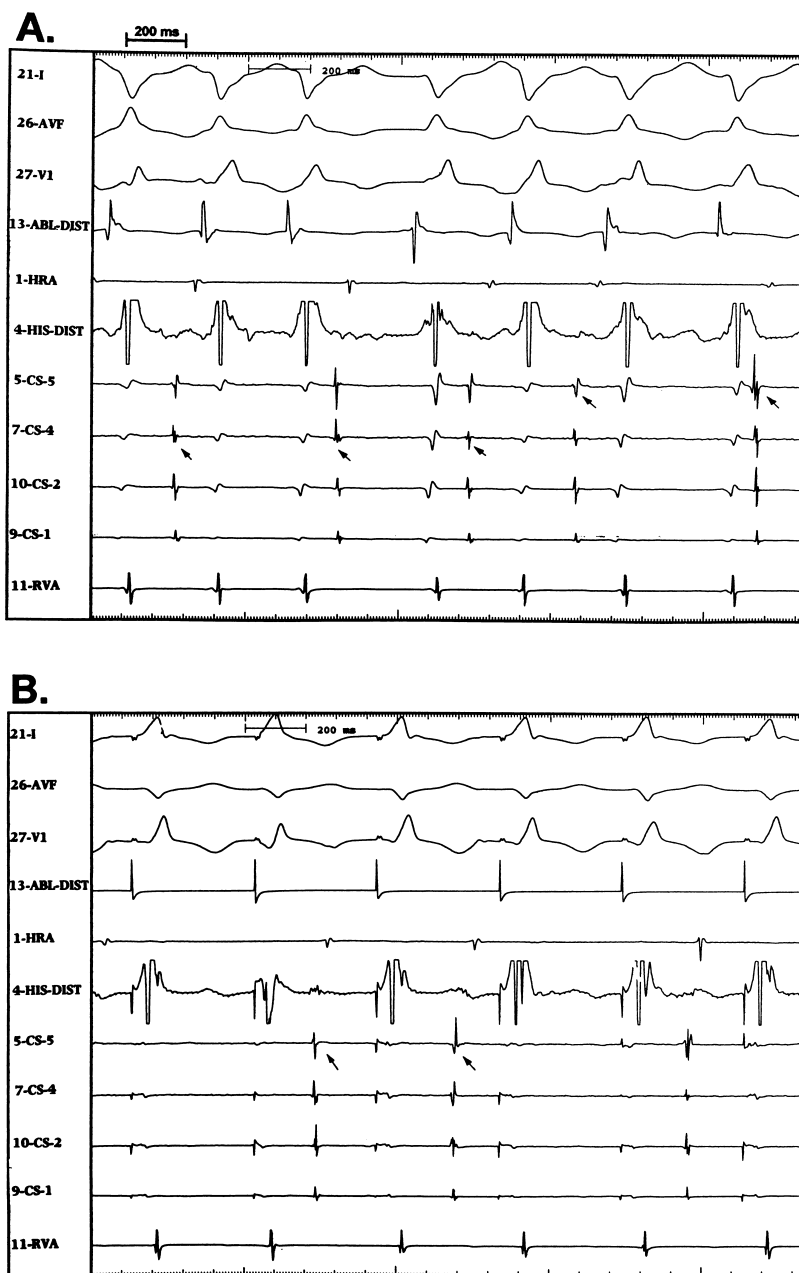
identified with eccentric retrograde atrial activation. In this patient and in two additional patients, the diagnosis of a left-sided AP was made during the study based on the presence of eccentric retrograde atrial activation sequences. Therefore, the left ventricular free wall region first received radiofrequency energy applications targeting the earliest retrograde atrial activation sites. No AP or SP potentials were recorded in these sites. All energy applications failed to terminate the RT or prevent its induction. Later during the study both typical and atypical AVNRT were induced. Slow pathway ablation was then performed according to standard protocols, with the ablation catheter positioned in the posterior region of the right atrial septum. After ablation, no RT was inducible with and without isoproterenol infusion. Because of this experience, in subsequent patients the radiofrequency energy was applied only in the SP region. Successful SP ablation prevented the induction of RT in all remaining patients.

In three patients, a typical slow-fast form of AVNRT was induced only after the successful SP ablation that terminated the slow-slow AVNRT. In these patients, the second SP was successfully ablated at a different site in the right atrial septum. If we look at the CS from a 45° right anterior oblique view, the most common sites of successful SP ablation were located between the 3 o'clock position of the CS orifice and the tricuspid annulus. In all 20 patients, SP ablations resulted in the abolishing of all forms of RT. None of the patients received radiofrequency energy application inside the CS.

Discussion

Eccentric retrograde atrial activation sequence. Of all methods used to localize APs, Gallagher et al. (23) found the sequence of retrograde atrial activation recorded during ventricular pacing and during RT to be most helpful. This

Figure 4. Eccentric retrograde atrial activation sequences during left ventricular pacing in the same patient as that in Figures 2 and 3. **A**, VA Wenckebach with varying degrees of eccentricity of retrograde atrial activation during short runs of ventricular tachycardia induced during manipulation of the ablation catheter in the left ventricle. **B**, Retrograde atrial activation sequences during left ventricular pacing with a cycle length of 500 ms, obtained after successful SP ablation. Other abbreviations as in Figure 2.



hypothesis was based on the observation that the normal sequence of retrograde atrial activation (23) started from the orifice of the CS, on the atrial septum near the His bundle, or was simultaneous at these two sites. Compared with this normal sequence of retrograde atrial activation, the presence of eccentric retrograde atrial activation is highly suggestive of the presence of an AP.

In this study, however, we demonstrated that 20 (6%) of the 356 patients with AVNRT had eccentric retrograde atrial activation sequences during RT. In 12 of the 20 patients, the earliest retrograde atrial activation occurred in bipolar pairs 3 and 4, compatible with the activation near the left side of the septum. Similar results have been reported by other investiga-

tors (25,32). However, in 8 of the 20 patients in the present study, the earliest retrograde atrial activation during RT was registered by the distal CS electrodes. An alternative explanation for eccentric retrograde atrial activation during AVNRT is the presence of a bystander left-sided AP. However, this explanation is not supported by the absence of VA conduction during the baseline pacing in 6 of the 20 patients and the presence of decremental conduction due to the HA prolongation during ventricular pacing in all patients.

This phenomenon has not been previously reported and could result in the misdiagnosis of the mechanisms of RT. The advent of radiofrequency ablation techniques have significantly increased the number of patients undergoing electrophysi-

ologic studies for supraventricular tachycardia. The increased case load may have helped us to identify a small subset of patients with unusual electrophysiologic characteristics. Based on the results of this study, both AVNRT and AVRT may be associated with an earliest retrograde atrial activation in the left atrium. Therefore, other methods of validation (23) must be used to demonstrate the presence of an AP before applying radiofrequency energy in the left AV junction in these patients.

The mechanisms by which eccentric retrograde atrial activation occurs during AVNRT are unclear. The most likely cause is that the perinodal transitional cells (18–20) documented in the histologic studies are not electrically silent and serve as an atrial input to the compact node. In a recent study of an animal model McGuire et al. (33) demonstrated that cells around both AV annuli have electrophysiologic properties that resemble the AV node cells. These cells may form the substrate of the SP and serve as inputs to the AV node. During AVNRT, the electric impulse could find its way from the AV junctional region into the left atrial annulus through these cells. Depending on the site of insertion of these cells, the apparent sequences of activation recorded by the CS catheter

Figure 6. Eccentric retrograde atrial activation sequence during AVNRT in a different patient from that shown in Figures 2 and 3. **A**, Activation registered during sinus rhythm. The atrial electrogram on both the His bundle and the CS catheters showed proximal (prox) to distal (dist) activation sequences, compatible with appropriate catheter placement. The AH interval was 71 ms and the HV interval was 46 ms. **B**, Induction of sustained AVNRT. **Arrows** point to the earliest retrograde atrial activation, which was in the distal CS. Other abbreviations as in Figure 2.

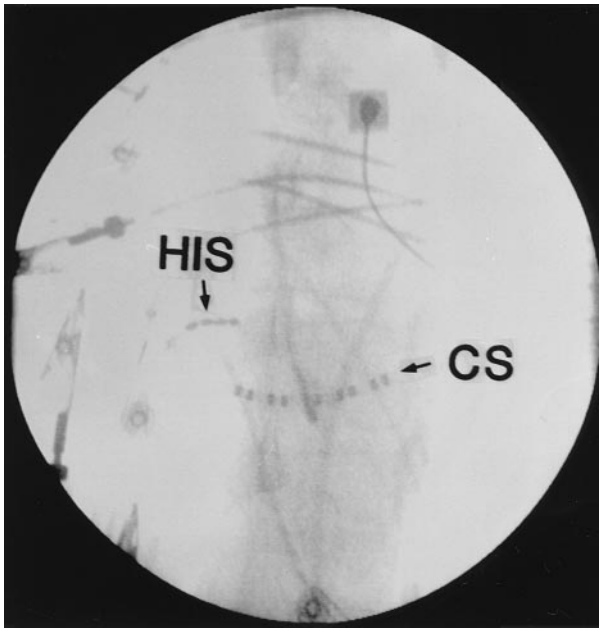
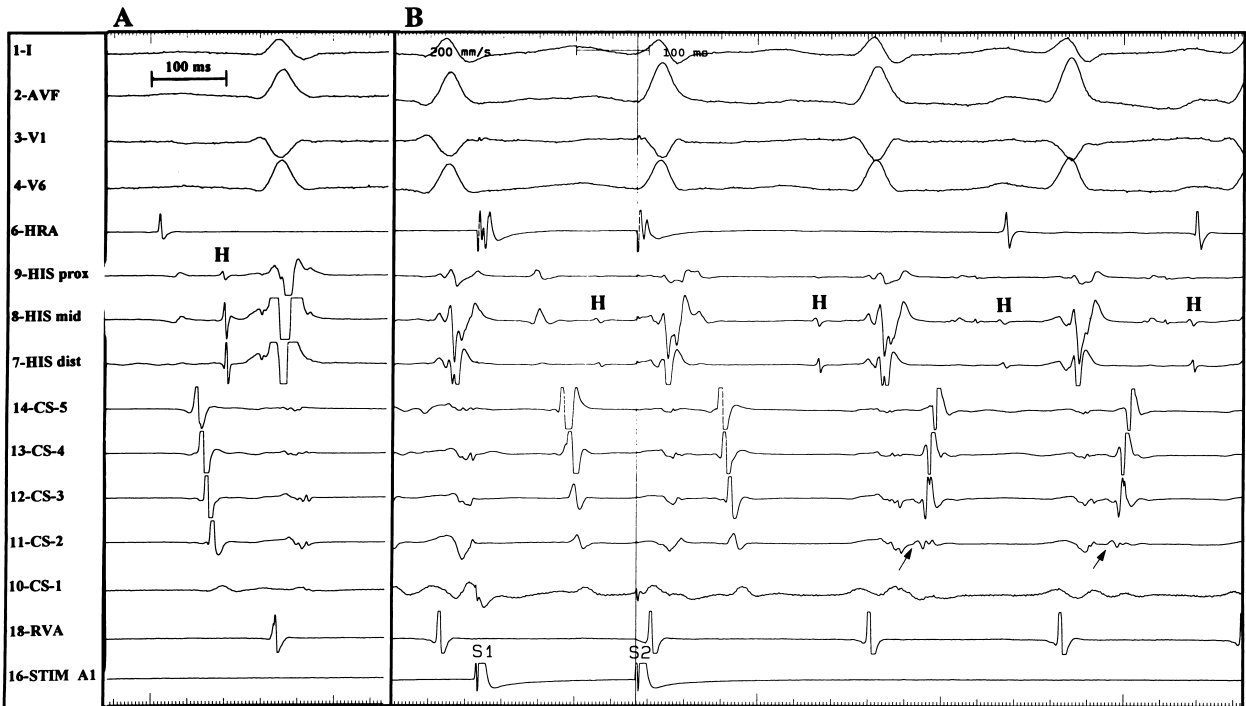


Figure 5. Fluoroscopic image of catheter location from the same patient as that in Figures 2 to 4, during the same study. This image (anteroposterior projection) was recorded by the Arrhythmia Research Technology CardioLab System. The His bundle electrodes (HIS) and the distal CS electrodes were far apart, indicating that the distal CS electrodes were in the left ventricular free wall location. Other electrodes were not clearly shown in this figure.

could be eccentric. These left atrial exit sites in some patients can differ during ventricular pacing and during RT, suggesting that anterograde and retrograde exit sites of these atrial inputs

were different. Whether these left-sided atrial inputs participate in the RT circuit is unclear. However, in three patients we have attempted radiofrequency catheter ablation near the CS electrodes that registered the earliest retrograde A wave. These attempts failed to eliminate the RT. In comparison, selective SP ablation successfully prevented the reinduction of AVNRT in all 20 patients. These findings indicate that the low right atrial septum served as part of the reentrant circuit, whereas the left atrium, which is distant from the ablation electrode during the SP ablation, was not critically involved in reentry.

If the eccentric retrograde activation is indeed due to the presence of AV node-like cells around the AV annuli, then it should be possible for some patients with AVNRT to have eccentric retrograde atrial activation sequence along the right AV annulus as well. However, because we did not perform mapping in that area, whether right-sided eccentric retrograde activations also occur in AVNRT is unknown.

Conclusions. Multiple SPs are present in the AV junctional region, and several different forms of AVNRT may coexist in some patients. In 6% of our patients the atypical form of AVNRT with eccentric retrograde left-sided activation sequences was demonstrated, masquerading as tachycardia using a left-sided accessory pathway. A cure of this form of AVNRT can be achieved by delivering radiofrequency energy at areas anterior or adjacent to the CS ostium without the need for ablation in the left AV annulus.

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